Extended Abstract

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Acute kidney injury presentation with recurrent hypoglycemia: A detailed case report of high-risk patients

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Abstract

Acute kidney injury (AKI) and hypoglycemia are independent risk factors for morbidity and mortality with a robust association. We present a case report of AKI related to hypoglycemia during a diabetic patient. Following standard AKI treatment protocol, kidney functions recovered, and therefore the patient had no further hypoglycemic attacks. AKI in diabetic patients might be multifactorial, but we propose the presenting symptom to the first care centre in our case was the recurrent hypoglycemic attacks which may have been overlooked by clinicians.While the association is well established in hospital settings, presentation in medical care settings with hypoglycemia is usually overlooked. This case report aims to spotlight the danger for AKI in diabetic and elderly patients with multiple risk factors for.

Introduction

Acute Kidney injury (AKI) is an abrupt reduction in renal functions presenting with the deterioration of one marker or more of the urea, creatinine, or urine output. The term encompasses an entire spectrum from minor injuries to end-stage renal disease.Prerenal disease alone accounts for up to 75% of the presentations while renal and post-renal diseases cause the rest.The prerenal term refers to a decrease in renal perfusion that could be caused by volume depletion, oedematous states, selective renal ischemia, or medications. The severe prerenal disease could lead to acute tubular necrosis (ATN). ATN term encompasses other pathological necrotic manifestations like endothelial dysfunction, coagulation abnormalities, systemic inflammation, and oxidative stress.Urinalysis and recovery period may help distinguish between prerenal disease and more severe form of ATN injury. The urinalysis is normal or near normal in the former, whereas in ATN it shows muddy brown granular, epithelial cell casts, and free renal cells. Recovery of the serum creatinine to baseline occurs within 24 to 72 hours following fluid replacement with the prerenal causes; longer recovery is more suggestive of ATN. Both prerenal and ATN presentations could overlap and coexist in an intermediate syndrome.

Keywords

Case report, Hypoglycaemia, Acute kidney injury, Diabetes

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Discussion

The patient had multiple risk factors for AKI that has DM, NSAIDs and ARB (losartan). Of note, she had NSAIDs on multiple occasions and continues to have glimepiride with no effect of both on her kidney functions and her glycaemic control. ARB (losartan) induced AKI explains the episode; an alternative explanation would be the simultaneous administration of the three medications.The kidney functions recovered over ten months which is more suggestive of ATN. The urine sample was clear throughout, that's more implicational decreased renal perfusion. Our patient showed a picture that is conflicting and renal injury might have been that of intermediate syndrome.Medications cause AKI through different mechanisms and injury tends to happen 7 to 10 days after starting. NSAIDs and losartan could affect the glomerular hemodynamic and/or kidney functions. NSAIDs decrease the afferent arteriolar dilatation, and ACE/ARB is suggested to cause efferent arteriolar constriction.

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